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April 29, 1991

Scientific Officer Code: 1114SE
Dr. Clifford Lau
Office of Naval Research
800 N. Quincy Street
Arlington, Virginia 22217-5000 696-4961

Dear Dr. Lau:

Enclosed are three copies of the third semi-annual progress report for Grant No. N00014-90-J-1114, "Pulse Coded Biologically Motivated Neural-Type MOS Circuits." I believe that the progress continues to be good and that the involved researchers remain quite excited about their continuing research. Indeed there are many graduate students here anxious to join in this research. Consequently, I continue to thank you for your encouraging support.

Please do let me know if further information is desired.

Sincerely,

Robert W. Newcomb
Professor

RN/lc
Enclosure: Semi-Annual Report (3 copies)
cc: a) Researchers
b) Dr. Alan Craig (1 copy)
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Progress Report for Grant ONR N00014-90-J-1114
"Pulse Coded Biologically Motivated Neural-Type MOS Circuits"
For the Period 11/01/90 - 05/01/91

1. Introduction

This project has two aspects, one for ONR and one for AFOSR. The ONR portion is devoted to obtaining hardware implementations for the physiological representations used in the program SYNETSIM developed byy the neurophysiologist Dr. D. Hartline of Bekesy Laboratories. The AFOSR portion is for evaluation capabilities of the pulse coded philosophy of neural networks.

During the period covered by this report a number of results have been obtained, the following being the main accomplishments. On the ONR portion of the research the DRIVER module of SYNETSIM has been realized by a ideal electronic components and a design has been sent for chip fabrication of the neural arithmetic unit, based upon SYNETSIM pools. Another significant result is a technique developed, based upon SYNETSIM, to implement long term potentiation. On the AFOSR portion, the pulse coded neuron which gives all logic functions has been improved for the possibility of construction via integrated circuits and a technique to implement any Hopfield-type network via pulse coded circuits initiated. Based upon the results for this Grant, one Ph.D. and one MS thesis have been presented.

Participating in the research during this period have been:

a. R. Newcomb, PI
b. N. El-Leithy, Co-PI
c. S. W. Tsay, RA - ONR portion
d. M. de Savigny, RA - AFOSR portion

In addition several other graduate and undergraduate students have taken independent study projects on related topics which enhance the research.

2. Research Results for the Period

2-A. ONR Aspects

In order to design further integrated circuits which mimic the compartments of SYNETSIM we have delved further into the operation of SYNETSIM. This has allowed us to isolate the DRIVER module which gives slow potentials of importance to setting threshold levels for spiking. During this investigation we have also run into the limitations of SYNETSIM in that a number of its functions are not yet implemented or have bugs. Consequently, some effort has gone into a further understanding of the concepts so that eventually more can be accomplished with SYNETSIM and true modules can be developed for integrated circuit realization. This should also lead to a vastly improved software program. At this point we do have the DRIVER module simulated in a functional form which we next plan to design via MOS transistor circuits. Along with this, a post inhibitory rebound structure has been designed which it is planned to eventually implement with CMOS circuits. Another aspect of this research which we have decided to pursue is the development of circuits which will realize long term potentiation, LTP. In some rather fascinating research, a schema has been developed for realizing LTP using ideas stemming from SYNNETSIM and our former neural type microsystems.

2-B. AFOSR Aspects

During this period improvements have been made in the pulse coded neuron which realizes all 16 binary valued logic functions of two inputs. Because the original circuit is inconvenient for VLSI, a study has been made of how to delete the filters used in the previously reported neuron. From this we have been led to a new pulse coded neuron which shows promise for realizing any Hopfield type of neural network in the pulse coded framework. At this point the new pulse coded neuron has been shown to allow us to also generate 14 of the 16 binary valued logic functions without the use of the inconvenient filters of the previous pulse coded neuron. Also using the new neuron a two input MAXNET has been realized. A major problem in implementing Hopfield types of neural networks in the pulse coded philosophy is how to encode general weights. A promising technique to solve this problem is under investigation.

3. Publications and Theses

A. Publications

During this period the first of the following papers was published and the others accepted for presentation at the respective meetings. Several other papers are in the final stages of preparation.

a) S. W. Tsay and R. Newcomb, "VLSI Implementation of ART1 Memories," IEEE Transactions on Neural Networks, Vol. 2, No. 2, March 1991, pp. 214 - 221.

b) S. W. Tsay, M. de Savigny, N. El-Leithy, and R. Newcomb, "An all MOS Neural-Type Cell," 34th Midwest Symposium on Circuits and Systems, 1991, to appear.

c) S. W. Tsay and R. Newcomb, "A Neural-Type Pool Arithmetic Unit," Proceedings of the IEEE International Symposium on Circuits and Systems, Singapore, June 1991, to appear.

d) G. Moon, M. Zaghloul, M. de Savigny, and R. W. Newcomb, "Analysis and Operation of a Neural-Type Cell (NTC)," Proceedings of the IEEE International Symposium on Circuits and Systems, Singapore, June 1991, to appear.

B) Theses

The following two theses, which were finished in April 1991, are based upon research under this Grant.

a) S.-W. Tsay, "Design of Neural Chemical Pools for VLSI and Their Applications," Doctoral Dissertation, University of Maryland, finished April 1991 for May graduation.

b) M. de Savigny, "Pulse Coded Neuron Realizing Digital Functions," MS Thesis, University of Maryland, finished April 1991 for May graduation.

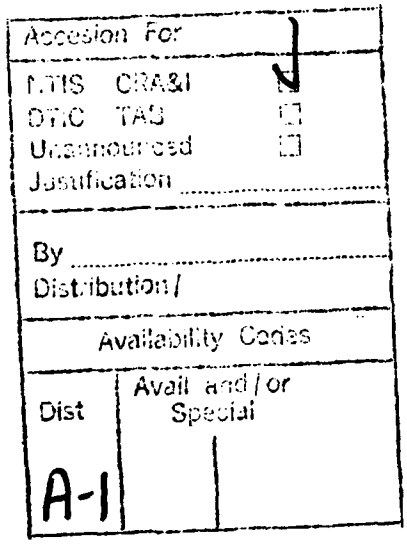
4. Research Assistant Reports

Attached are concise reports from the two Research Assistants and the Faculty Research Assistant (Co-PI) for the Grant.

5. Plans for Next Period

We plan to improve the capabilities of SYNETSIM by making it more modular and correcting some bugs. This will also allow us to design more CMOS circuits to go with the stand alone modules. Some chips have been sent out and we hope to have these back in working order so that testing can proceed which in turn could lead to further improvements.

It appears that added functions can be realized by improvements in the neural arithmetic unit and research to do that is planned. A major effort will go into a further development of the theory for LTP, its realization in SYNETSIM form, and its design in MOS circuits. Because of the significance of realizing Hopfield type neural networks in pulse coded form, further effort will be devoted to that aspect of the research.



Dist. A. per telecon Dr. C. Lau
ONR/Code 1115 SE

CG 5/08/91

Research Report

Research Assistant: Suan-Wei Tsay

Our research works have emphasized on four aspects during the period from 11/1/90 to 5/1/91. These are 1) Circuit realization of DRIVER module, 2) Design the post inhibitory rebound scheme using chemical pools of type 1, 3) A paper titled "A Neural-Type Pool Arithmetic Unit" is accepted and to appear by the Proceedings of the IEEE International Symposium on Circuits and Systems, Singapore, June, 1991, and 4) A Ph.D. dissertation based on the research supported by this grant is finished. Parts 1) and 2) are discussed below:

1. Circuit Realization of DRIVER module

The driver module is a circuit model of neuron membrane to the depolarization and hyperpolarization of the membrane potential. The circuit of DRIVER is shown in Fig. 1.

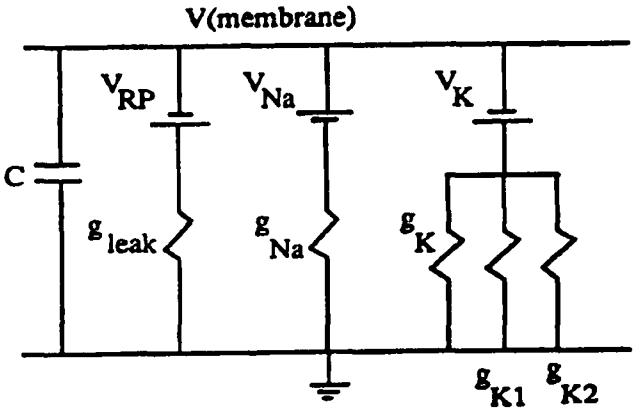


Figure 1: The circuit for DRIVER to cause depolarization and hyperpolarization on membrane potential.

In Fig. 1, two conductances, g_{Na} and g_K, are voltage dependent with dependency on the membrane potential only. Two other conductances, g_{K1} and g_{K2}, however, are time dependent with dependency on both membrane voltage and time. The formulas for these conductances are

$$g_{Na} = \frac{\bar{g}_{Na}}{e^{(V_{0Na} - V_m)/\mu_{Na}} + 1} \qquad g_K = \frac{\bar{g}_K}{e^{(V_{0K} - V_m)/\mu_K} + 1} \qquad (1a,b)$$

$$\frac{dg_{K1}}{dt} = \left(\frac{\bar{g}_{K1}}{e^{(V_{0K} - V_m)/\mu_K} + 1} - g_{k1} \right) / \tau_{k1} \qquad \frac{dg_{K2}}{dt} = \left(\frac{\bar{g}_{K2}}{e^{(V_{0K} - V_m)/\mu_K} + 1} - g_{k2} \right) / \tau_{k2} \qquad (1c,d)$$

where $\bar{g}$.'s are maximum conductances, V_m is the membrane potential. V_{0Na}, V_{0K}, V_{Na}, V_k, τ's, and μ's are all constants. The PSPICE simulation of this driver circuit is shown in Fig. 2.

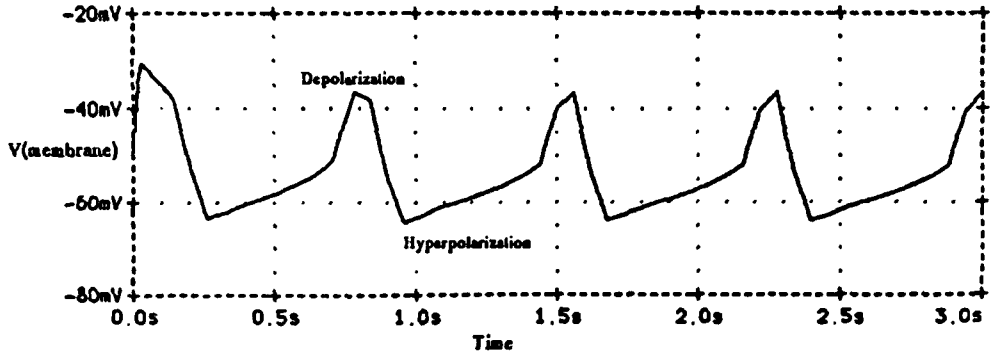


Figure 2: PSPICE simulation output for DRIVER circuit.

Two DRIVERs can be connected together by a coupling resistor so that their depolarization-hyperpolarization frequencies can be synchronized. Figure 3 shows two drivers with a coupling resistor. When the coupling resistor is taken off, these two drivers are with different frequencies due to the different choice of $\tau_{K's}$ in DRIVERs 1 and 2 (Figure 4(a)). Figure 4(b) shows outputs for these two DRIVERs that their output are synchronized due to the coupling conductance.

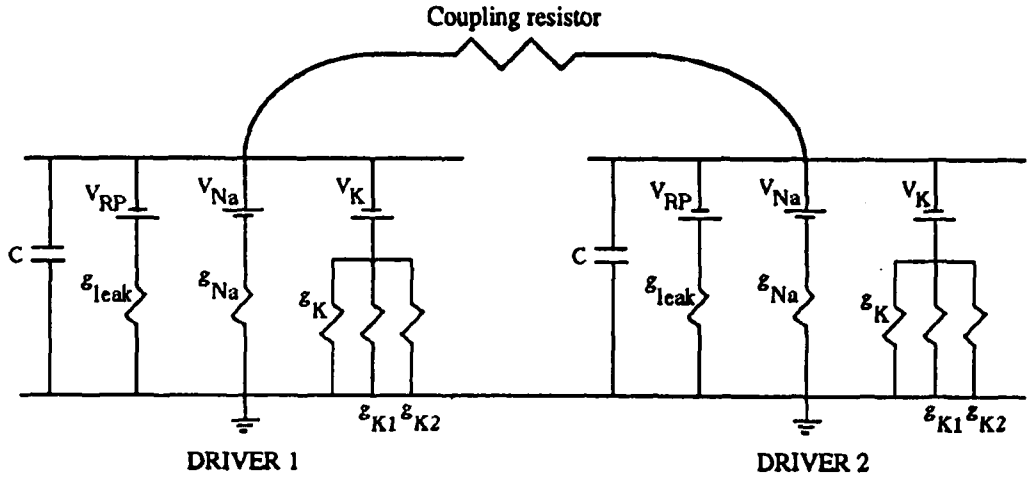


Figure 3: Two DRIVERs with a coupling resistor.

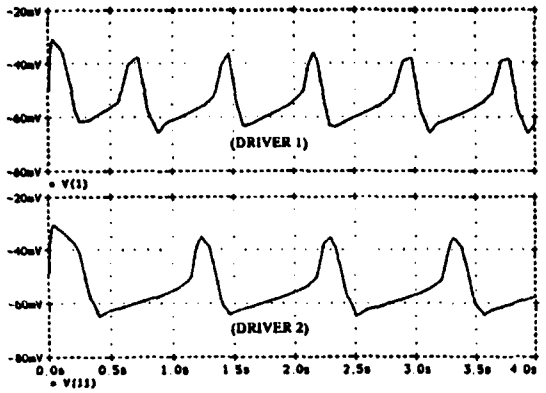


Figure 4(a): PSPICE simulation for 2 DRIVERs with no coupling resistor.

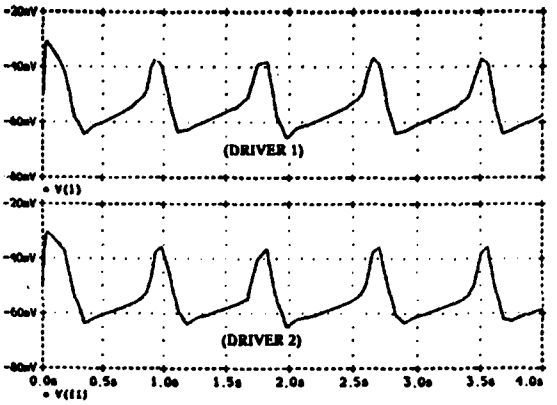


Figure 4(b): PSPICE simulation for 2 DRIVERs in Fig. 4(a) with coupling resistor.

2. Post Inhibitory Rebound

Post inhibitory rebound can be realized by the circuit of Figure 5 where P_a and P_b are type 1 chemical pools acting as bound transmitters. E is the excitatory input and I is the inhibitory input. The pool levels of P_a and P_b depend on the states of neurons A and B, respectively. That is, $[P_a] = \frac{M}{N + x_a}$ and $[P_b] = \frac{M}{N + x_b}$ where M, N are positive constants and

$[\cdot]$ represents the pool level (concentration of pool material). When I > E, the output is negative. Suppose the inhibitory signal I is taken off immediately, due to the slow recovery rate of the pool level, the output will rebound to positive. The complete CMOS implementation is still under investigation.

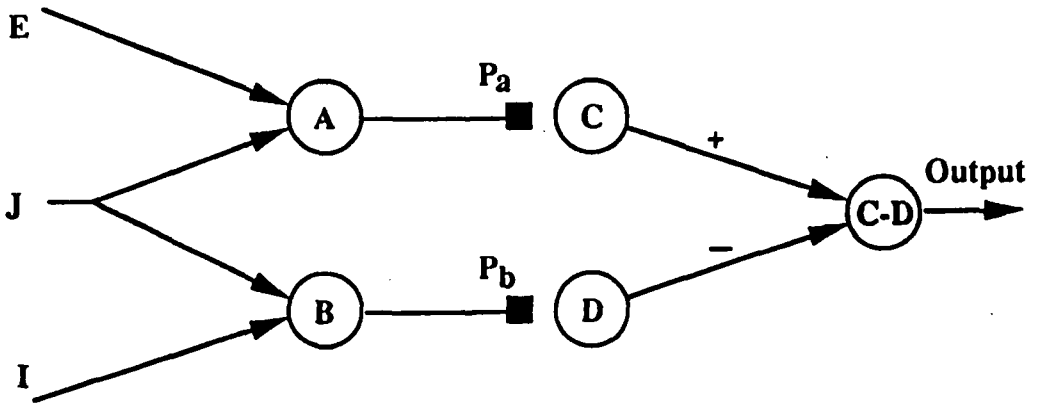


Figure 5: Circuit diagram for post inhibitory rebound.

Research Report

Research Assistant: Marc de Savigny

Summary:

Research performed during the period from 11/1/90 to 5/1/91 has included additional work on the pulse coded neuron presented in the previous research report, and study of a pulse coded Hopfield neural network. A Master of Science Thesis has been written based on the research supported by this grant.

The pulse coded neuron:

In the last months, we have found electrical circuits for the different elements of the pulse coded neuron. Of course, it still suffers from its major drawback: it is fairly complex and should occupy a large area on a chip. This jeopardizes its use in a real application, since not so many neurons can be integrated on the same chip.

It appears that the pulse coded neuron we have studied is an existence proof that Boolean functions of 2 variables can be implemented with neurons. A very interesting and far-reaching consequence is that all the digital machines conceivable can be implemented using artificial pulse coded neurons. This is because any logic machine can be theoretically decomposed in a combination of elementary gates plus some memory elements. A pulse coded memory cell was developed to conclude this study.

Hopfield pulse coded neural network:

Our new aim is to show that any present day neural network can be implemented with pulse coded neurons. As a first step, we consider the classic structure of Hopfield neuron networks where the neurons have been replaced by pulse coded ones. In order to simplify further, Boolean functions of the network's inputs are studied. In such a network, the outputs would be either a constant zero level, or pulses if the corresponding neuron spikes.

For this, we will use the NTC as the pulse generating element. A device called "cell", placed at the input of the NTC takes the signals from one input to the network and the output signals of all the other neurons, to combine them. The result determines whether the NTC under consideration oscillates or not. Our main constrain this time is to get a small circuit. VLSI implementations of the NTC have been studied in the past by Mr Tsay, and will not be done here. The cell is therefore what we need to concentrate our design efforts on.

Finally, a 3-neuron pulse coded Hopfield network has been created. The network performs the function "Biggest of 2 inputs", when the third is set to the supply voltage, and "2 inputs are pulses", when the third is set to the ground. Minor modifications of the inputs to the network allow to perform 14 of the 16 Boolean functions of 2 variables. The 2 functions left out are "exclusive or", and "not exclusive or".

Future research:

Additional research on the pulse coded Hopfield network needs to be done to evaluate its capabilities better. We are currently working on how to be able to accept pulses as inputs to the network versus DC levels like it is now done. An other issue is to design a system to change the weights of the network easily. A small network could then be integrated and tested in a chip.

RESEARCH REPORT

Co-PI: Nevine El-Leithy

FOCUS:

Naturally as one navigates through neuroscientific disciplines and their allies, the temptation to understand the roots of cognition becomes irresistible. Intrigued by the physiological accompaniments of learning and memory, we proceeded to investigate the possibility of using Hartline's approach, as modeled by the SYNETSIM series, to isolate and trace generic membrane-bound electrobiochemical mechanisms that can define the 'how' and the 'when' of learning and of memory consolidation. We focused on the extremely appealing phenomenon of hippocampal long-term potentiation (LTP) as a model form of neuroplasticity that could possibly underlie associative learning. The goal is to use the compartments and pools of SYNETSIM to capture the molecular and cellular basis of LTP and then proceed to electronically implement an LTP-based cognitive trace. Such a process required: first, mastering the "figures of speech" of the relevant neuroscientific literature in order to develop our own engineered syntax, and, then, relating the resulting semantics to Hartline's SYNETSIM model.

THE PROPOSED SCHEME:

Integrating the crucial information abstracted so far, the schematic picture of Figure (I) emerges as the neural substrate of the proposed LTP-based associative model. In this model, two afferent fibers 'a' and 'b' synapse on two neighboring spines 'A' and 'B', respectively. Both spines contain excitatory amino-acid [EAA] receptors as well as voltage-dependent channel mechanisms. Spine A head membrane is assumed to comprise a K/Q receptor-channel complex, two voltage-dependent channels: (1) a fast calcium current I_{CaA}, (2) a slow, noninactivating potassium current I_{MA}, similar to that reported in hippocampal pyramidal cells, and (3) a calcium-dependent voltage-independent potassium current $I_{K(Ca)}$ causing long hyperpolarizing pauses and reported in almost every excitable cell. I_{CaA}, which is similar to that found in the hippocampus and in other locations, is believed to underlie the high-threshold Ca^{2+} spikes most likely originating in the dendritic tree and is assumed to become inactive with increased levels of intracellular calcium, $[Ca^{2+}]_i$.

Spine [B] head membrane is assumed to comprise a K/Q receptor-channel complex and an NMDA receptor-channel complex. In addition, it contains the slow, noninactivating potassium current I_{MB}, as well as, two calcium-dependent potassium currents: (1) I_{AHP}

which is voltage-independent, and (2) I_c which is noninactivating and its activation is a function of both $[Ca^{2+}]_i$ and membrane voltage. Signals from both spines connect through a dendritic shaft whose membrane is assumed to contain a $GABA_A$ receptor-channel complex and a transient outward potassium current mechanism I_A which strongly affects responses to hyperpolarizing current injections. Both spines are assumed to include calcium-buffering systems operating at different binding rates.

The spine [A] system relays strong signals to its neighboring dendritic patch in response to stimuli approaching its presynaptic terminal. On the other hand, spine [B] is assumed to initially relay weak signals to its neighboring dendritic patch in response to stimuli approaching its presynaptic terminal. However, after repeated exposures to specific patterns of stimuli approaching both presynaptic terminals almost concurrently, the membrane structures of spine [B] respond by relaying stronger messages to the underlying dendritic shaft. LTP is assumed to have become induced, formulated and expressed in the infrastructure of spine [B].

The scheme of Figure (I) can be electrically modeled following Hartline's philosophy as shown in Figure (II). The intracellular modulatory events, which ultimately result in plastic changes within the spine [B] structure are, indicated by the pools and the pool interactions (circles and arrows) of Figure (II). The attempt is to capture the electrical behavioral modifications in spine [B] head membrane in terms of underlying ionic currents and the background activity of biochemical interactions.

The electrobiochemical events evolve over two time-scales: a primary-time-scale during which LTP is induced after the system has been bombarded by concurrent tetanic stimuli: [a] and [b], and a modulatory time-scale during which LTP is formulated and expressed. The induction process is generated postsynaptically as a result of the activation of the NMDA receptor and the opening of the Ca^{2+}-channel it controls. The NMDA receptor channel complex is represented by two electrical branches: $[g_{NMDA}, V_{NMDA}]$ and $[g_{CaB}, V_{Ca}]$. Two conditions are mandatory for such induction: binding of the endogenous ligand (glutamate) to the NMDA receptor, and a sufficient level of membrane depolarization to remove the magnesium blockade of the NMDA-Ca-channel. The inductive step in the genesis of LTP is the influx of Ca^{2+} into the spine [B] head membrane during tetanic stimulation after Mg^{2+} pops out to allow I_{CaB} to flow. Note that the level of depolarization of spine [B] head membrane is a function of its intrinsic ligand-and voltage-gated channels as well as the electrotonically transferred signal arriving from spine [A] head.

The entry of Ca^{2+} through the NMDA-Ca^{2+} channel provides the crucial link between electrical stimuli [a] and [b] and the initiation of intracellular events that result in LTP formulation and expression. In the proposed scheme, P_{CaB} represents the

build-up of intracellular $[Ca^{2+}]_{iB}$ in spine [B], which binds with a hypothesized calcium-dependent activator (CDA) forming the pool P_{CDA}. P_{CDA} is then responsible for the production and activation of a postulated "plasticity factor" from an inactive form (PF) into an active form (PF*). The putative PF* could then act at either or both sites: on spines postsynaptically, or, if released from dendrites into the extracellular space, on presynaptic terminals.

The postulation of a plasticity factor (PF) is general (or plastic!) enough to be used as part of any of the diverse mechanisms proposed by different laboratories to account for the sustained increase in synaptic efficacy that characterizes LTP. The working hypothesis shared by many laboratories is that Ca^{2+} serves as an intracellular (or second) messenger to trigger a sequence of events that would ultimately involve the postulated plasticity factor. The plasticity factor could represent any of the known protein kinases eliciting change by phosphorylating a membrane protein, or it could be one of the proteases causing change through the breaking or cleavage of phosphate bonds to cytoskeletal proteins. Moreover, the plasticity factor may represent an autophosphorylating kinase affecting cytosolic or genomic biochemistry thus storing information beyond the lifetime of any single protein, or it could act on contractile proteins, such as myosin and actin, causing rapid contractions or twitching of the spine neck.

The modulatory time-scale is divided into two time-zones: P_{CaB}, P_{CAD} AND P_{PF} are assumed to occur within an acquisition time-zone, while PF* and its relevant mechanisms occur within a so-called catalytic activity time-zone. PF* can act as a switch in that it can be converted to an activator-independent form, requiring neither Ca^{2+} nor CDA for catalytic activity. This catalytic activity of PF* should ultimately alter the electrical properties of spine [B]. LTP expression refers to the biophysical alphabet underlying the sustained increase in synaptic efficacy. Like LTP formulation, the site and manifestation of LTP expression are still controversial. In our model, four different mechanisms of LTP formulation are adopted: phosphorylation, cleavage, morphological changes and synthesis of a trophic factor, as shown in Figure (II). The ultimate expression is a function of which form or combination of forms of PF* is in action. The catalytic activity time-zone determines the life span of the expression.

Referring to Figure (II), 4 targets of modulation represent the syntax of the proposed scheme and are depicted as follows:

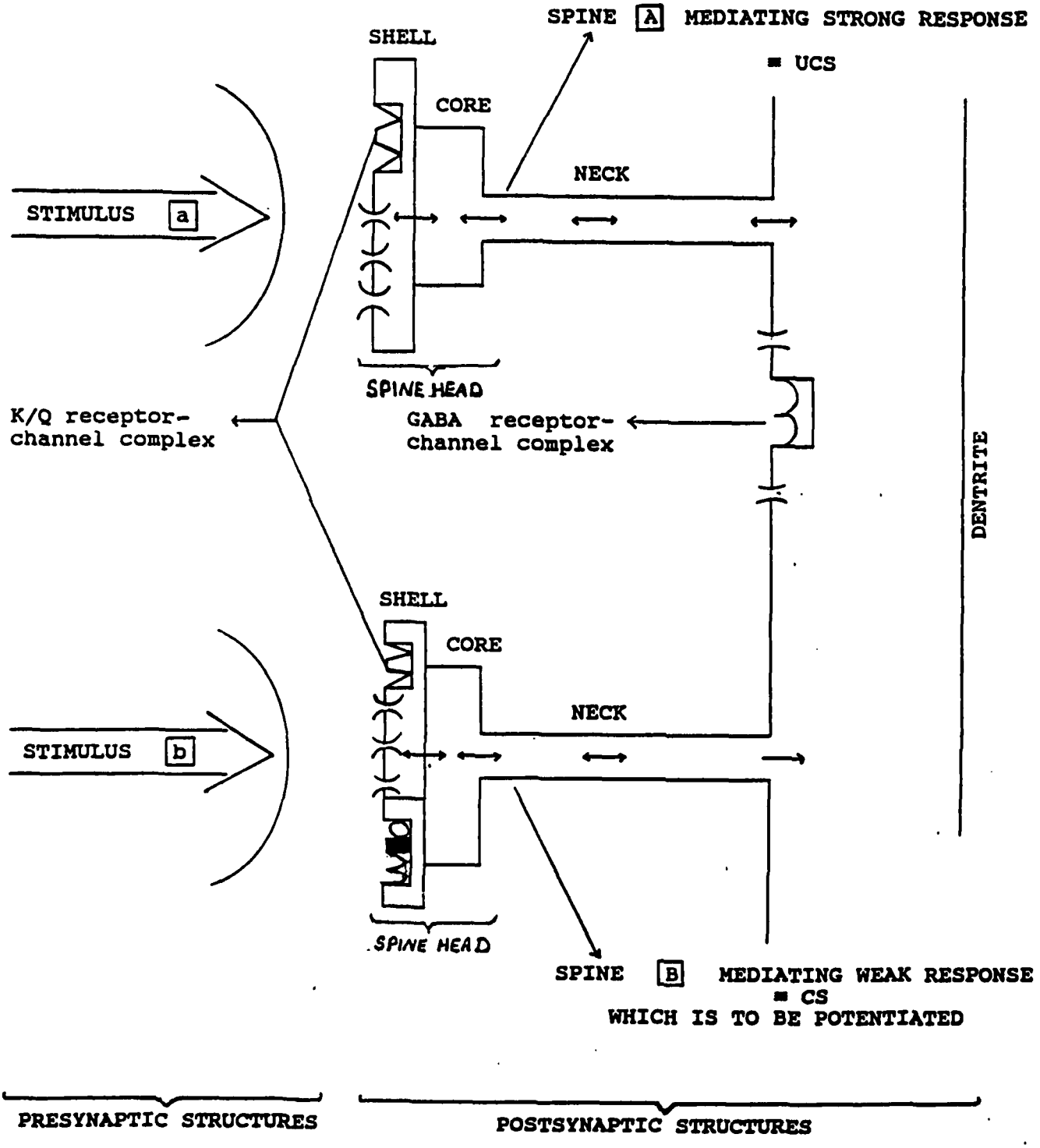
QE1	Reduction of calcium-dependent potassium currents of the conditioned spine: modulation of g_{AHP} and g_c.
QE2	Increase in the maximum postsynaptic conductance: modulation of g_{KQB}.
QE3	Reduction of the neck resistance of the conditioned spine: modulation of R_{NB}.
QE4	Enhanced release of glutamate from the presynaptic fiber of the conditioned spine: modulation of s(t) of [a].

The working assumption is that the above 4 alterations can occur simultaneously, independently, in certain combinations, or not occur at all (if spine [B] is not potentiated).

The expression of a 'learned' association will last for a long period of time determined by the catalytic activity time-zone.

FIGURE I

THE PROPOSED SCHEME FOR A HIPPOCAMPAL-TYPE ASSOCIATIVE MODEL



KEY

)(≡ channel

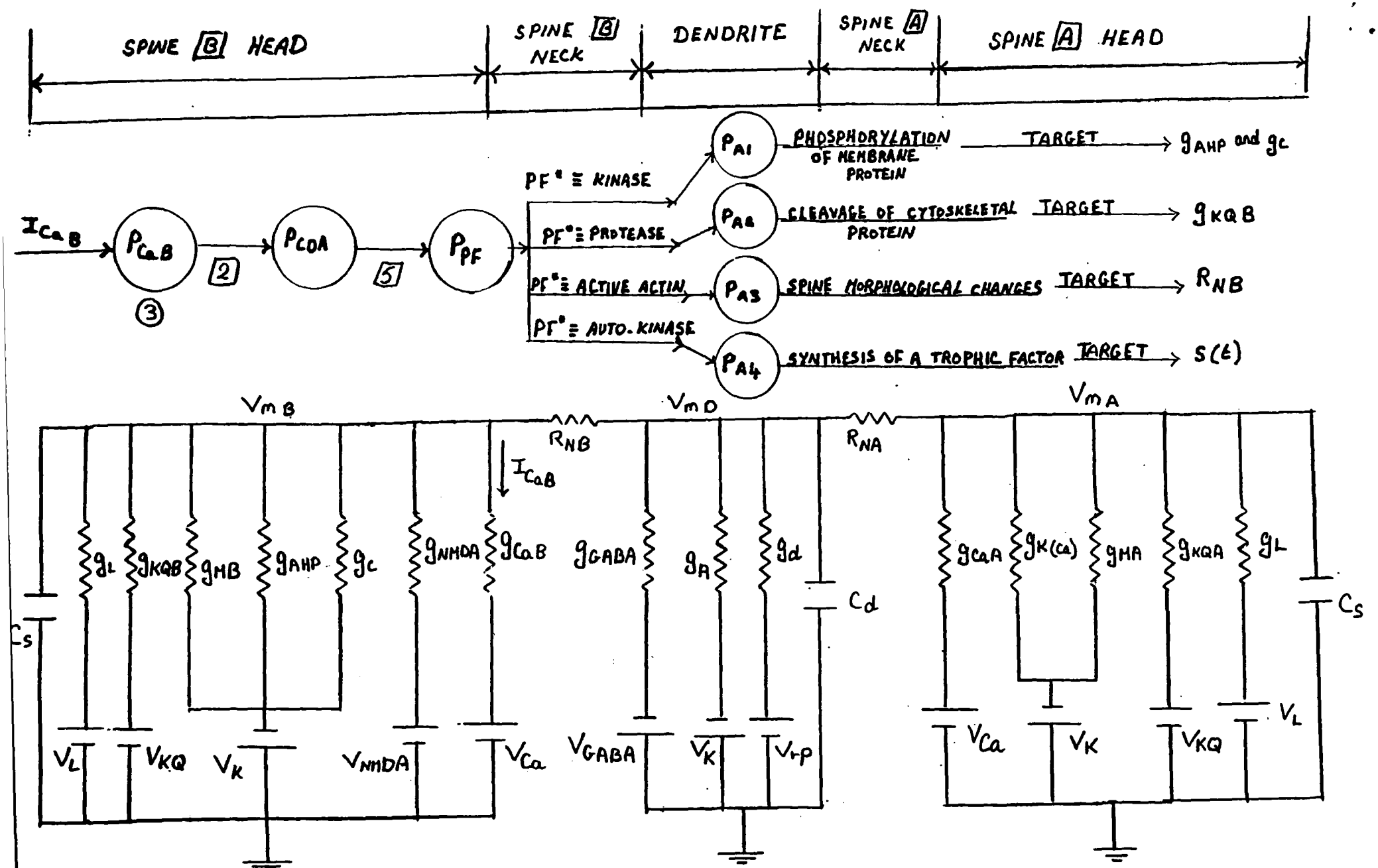
⟷ ≡ Ca^{2+}-diffusion

CDA: Ca^{2+}-dependent activator

Ca^{2+}-channel blocked with Mg^{2+}

≡ NMDA receptor-channel complex

→ NMDA receptor



ELECTRICAL REPRESENTATION OF THE STRUCTURE COMPRISING:
TWO SPINES CONNECTED BY A DENDRITIC SHAFT -
AND POOL REPRESENTATION OF BIOCHEMICAL EVENTS RESULTING
IN PLASTIC CHANGES OF ELECTRICAL PROPERTIES
OF SPINE B

FIGURE II